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Fluid-Dynamic Modeling of the Human Left Ventricle: Methodology and Application to Surgical Ventricular Reconstruction

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Background. **The efficacy of surgical ventricular reconstruction (SVR) for ischemic cardiomyopathy has never been truly quantified. Methods to assess ventricular flow have not been applied to these patients. The objective is to develop a volume-independent technique for assessing the effects of ischemic remodeling and SVR on left ventricular blood flow dynamics.**

Methods. **Cardiac magnetic resonance images from a healthy volunteer and from a patient before and after SVR were segmented and transformed to generate a grid model of the heart by generating numeric grids and running third-order approximations to achieve 850 grid images per cardiac cycle. These grids formed the skeletal structure of our patient-specific time-dependent ventricular geometry model, the Karlsruhe Heart Model, used for modeling fluid dynamics. We modeled flow, ejection fraction, and blood washout from the ventricle. The model was validated using a silicone ventricle and mock circulation.**

Results. **In the healthy heart and before SVR, ejection fractions were 0.61 and 0.15 and left ventricular volumes were 166 mL and 175 mL, respectively. Surgical ventricular reconstruction decreased left ventricular volume by one fourth. Postoperative ejection fraction was 0.18 in the patient. Post-SVR shape was more spherical than preoperatively and also more spherical than the healthy heart. Ventricular flow patterns in the patient were significantly altered by SVR. However, fluid washout from the ventricle was similar before and after SVR but worse than in the healthy heart.**

Conclusions. **Fluid dynamic modeling of the heart is possible based on cardiac magnetic resonance imaging data and enables volume-independent quantitative assessment of the surgical procedure. In the future, preoperative modeling for patients with remodeled ventricles may help to achieve optimized post-SVR flow characteristics and potentially outcomes.**

(Ann Thorac Surg 2009;87:1187–95)

The available options to treat patients with ischemic heart disease have undergone a tremendous development in recent years but they have not stopped the problem from growing. It is therefore necessary to use new strategies to improve the understanding of basic mechanisms as well as treatment options. We have used such a potential strategy by combining routine clinical diagnostic tools (magnetic resonance imaging) with a theoretical tool for fluid dynamic modeling to investigate the effects of a surgical strategy to treat patients with ischemic heart disease.

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Patients with ischemic heart disease frequently develop aneurysms or dilation of the ventricle in response to a regional infarct (remodeling) [1, 2]. Surgical ventricular reconstruction (SVR) or other modifications of the Dor procedure [3] have been advocated and used to treat patients with aneurysms or large akinetic anterior walls and dilated ventricles. The results seem convincing as evident by several reports on the short-term and long-term outcomes of this type of surgery [3–5]. Yet, despite the plethora of data already available, crucial aspects with respect to the efficacy of this procedure are still unanswered. A question that has stimulated heated debates is the optimal shape and size of the ventricle that is created by the reconstruction procedure. Buckberg and colleagues [6, 7] endorsed the concept that the creation of a football shape (ie, an elliptical ventricle with a cone-shaped apex) rather than a basketball shape (round ventricle without true apex) may provide the best base for optimal hemodynamics. The current controversy is

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difficult to solve as none of the diagnostic techniques applied so far has been able to truly quantify the effect of the procedure, either from a prognostic or from a hemodynamic point of view.

The goal of this study was to develop a volume-independent quantitative technique to assess ventricular flow dynamics based on fluid dynamic modeling and test the applicability of this methods to patients with ischemic remodeling and surgical ventricular reconstruction. In prototypical fashion, we used cardiac magnetic resonance imaging (CMR) data of a healthy human heart as input for the numeric model to simulate the blood flow through the heart. Two additional data sets from a patient with ischemic cardiomyopathy before and after SVR were also generated and analyzed in the model. Because this is the first effort to use this technique, we chose one patient and a healthy volunteer for this elaborate, time-consuming modeling process to establish a "proof of principle." We demonstrate that fluid dynamics can quantitatively be assessed by our method and that it is applicable in patients undergoing SVR.

Material and Methods

Human Subjects

One healthy volunteer and one patient were included in this investigation. The patient had ischemic cardiomyopathy and received coronary artery bypass graft surgery and SVR as indicated by the referring cardiologist and the surgeon and as randomized in the STICH trial [8]. Cardiac magnetic resonance imaging was obtained once in the healthy volunteer. In the patient, CMR was obtained before and 1 week after surgery. The patient received routine preoperative and postoperative care, and the CMR tests were performed in conjunction with the STICH trial. Both individuals agreed to participate in the study. The ethics board of the University of Freiburg approved the study, and the study conforms with the Declaration of Helsinki.

Magnetic Resonance Imaging and Echocardiography

Data acquisition was gated to the cardiac cycle and time-resolved (CINE) anatomic images were collected to depict the dynamics of myocardial motion during the cardiac cycle. To accomplish this task the measurement had to be repeated over a number of electrocardiographic cycles to gain a sequence of images representing different time frames of cardiac motion. All studies were carried out on a Siemens Sonata 1.5-T system (Siemens Medical Solutions, Erlangen, Germany; gradient performance: 40 mT/m in 200 μs) using an eight-element phased-array body coil. All measurements were performed with a retrospectively cardiac-gated balanced radio-frequency–spoiled gradient echocardiographic sequence with echo time/repetition time (TE/TR) = 2./3.4 ms and a bandwidth of 450 Hz/pixel. The flip angle was set to 15°. With nine echocardiograms acquired per cardiac frame and a data matrix of 126 × 192 (325 × 400 mm field of view; pixel size 2.6 × 2.1 mm), a temporal resolution of 30.6 ms was achieved. Images were acquired in a short-axis orientation with a slice thickness of 5 mm.

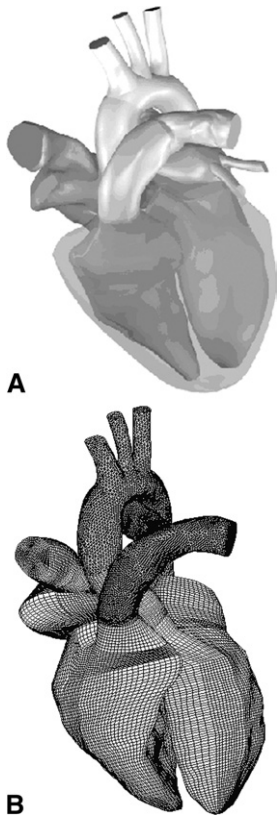

Fig 1. Schematic drawing of the Karlsruhe Heart Model (KaHMo; A) and the numeric grids obtained based on cardiac magnetic resonance imaging on a healthy volunteer (B). See Methods for description of grid generation.

Echocardiography with Doppler tracings were obtained from the patient as part of the routine preoperative and postoperative workup or additionally in the healthy volunteer to obtain model-relevant information on valve opening (see heart valve model below for details).

Karlsruhe Heart Model

The KaHMo (Karlsruhe Heart Model) [9–11] is a patient-specific numeric model of the human heart. It was developed to potentially aid the diagnosis and therapy of human heart diseases. Within the model, the heart is divided into an active and a passive part. The active part consists of the left and right ventricles as well as the left and right atria. Their movements were obtained from the segmentation data of the magnetic resonance studies and prescribed in the simulation. The passive part of the heart consists of the aorta, the venae cavae, and the pulmonary artery. This information was also obtained from magnetic resonance images. The information for the cardiac valves was obtained from echocardiographic Doppler images (see above). Figure 1A shows the sketch of the KaHMo. The segmentation of the CMR data was

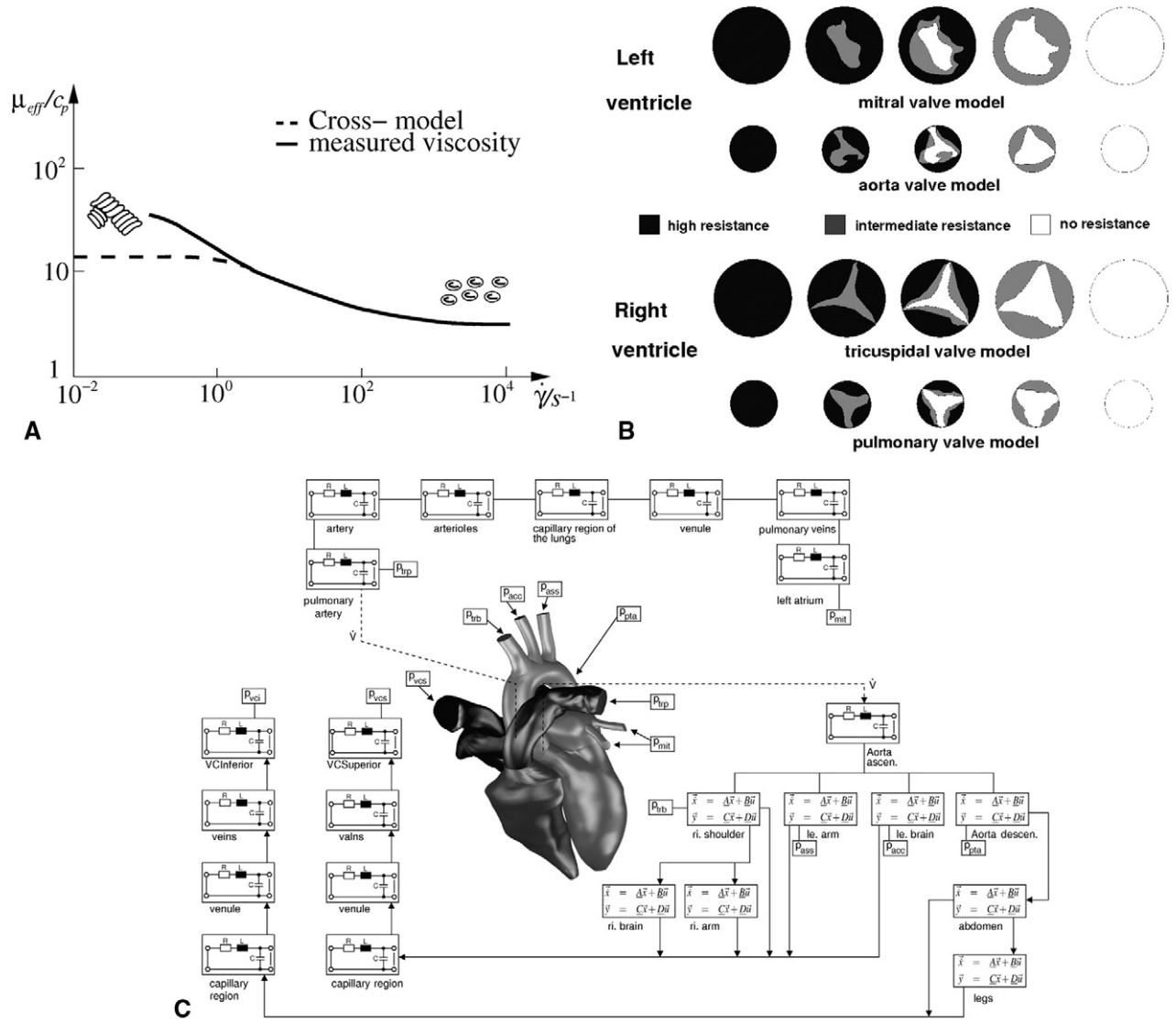

Fig 2. Schematic illustrations of the basic principles of blood viscosity (A), heart valve opening (B), and whole body circulation (C) as they are used for the modeling process. See Methods for details. (ascen = ascendent; descend = descendent; le = left; ri = right; VC = venae carae.)

performed by a semiautomatic live wire method provided by the Fraunhofer Institute for Applied Information Technologie (FIT) [12] that determines the endocardium contour in each magnetic resonance imaging slice. From these slices the inner surface of the ventricle can be remodeled for grid generation.

Grid Generation and Movement

The presented numeric model of the human heart is a prescribed geometry model using a moving grid without modification of the grid topology. It is built for each trigger step from the CMR using a semiautomatic grid generation technique as follows: a tubelike base grid is projected onto the segmented surface resulting in identical vertex distributions adapted to the individual geometry. The fluid space of the ventricle is covered with hexagonal cells arranged in two O-grids for the inlet and outlet tracts, which are combined and reach the ventricular apex. Figure 1B shows a representative grid from the numeric model of the healthy heart. From grid-dependency studies, 10^5 cells are sufficient to capture the global flow structures. For numeric reasons it is important to keep the Courant number close to unity. With the actual temporal resolution of the magnetic resonance imaging, this cannot be ensured. Thus, out of the existing grids intermediate grids had to be calculated. For this issue a twice differentiable third-order Bezier-Spline approximation method for every node of the grid was used, and 50 additional grids are approximated between two adjacent grids obtained from the CMR data segmentation. Thus, a total of 650 to 850 grids per data set (for one cardiac cycle) were used for fluid dynamic modeling with KaHMo.

Blood Model

Because of its composition of blood plasma and deformable blood corpuscles, the rheologic behavior of blood can be described as a mainly pseudoplastic, thixotropic suspension with Newtonian behavior for low and high shear rates and a shear-thinning behavior. Figure 2A illustrates the dependence of the viscosity (μ_{eff}) of the blood on the shear rate. The model used for the numeric calculations of the pulsing blood flow was based on the Cross model with modifications by Perktold and associates [13]. For a three-dimensional flow the viscosity-shear relation is as follows:

$$\frac{\mu_{II_d} - \mu_\infty}{\mu_0 - \mu_\infty} = \frac{1}{\left(1 + \left(2 \cdot \lambda \cdot \sqrt{II_D}\right)^b\right)^a}$$

The constants $\mu_0 = 0.1315$ Pa · s and $\mu_\infty = 0.03$ Pa · s describe the asymptotic viscosity for low and high shear rates, respectively. II_D is the second invariant of the shear rate tensor. The time constant $\lambda = 0.5$ s and the model constants $a = 0.3$ and $b = 1.7$ are adapted from the experiments of Liepsch and colleagues [14].

Model of Heart Valves

To simulate the whole cardiac cycle, valve function has to be implemented into the numeric model. The valves are represented by a two-dimensional annular planar model. Onto this plane, an opening area, derived from echocardiographic Doppler scans, is projected. Numerically, the valves are represented by a temporally and spatially variable pressure drop, realized by so-called baffle boundary conditions. The pressure drop was varied from infinite (closed valve area) to zero (open valve area). An intermediate pressure drop was defined for the border of the opening area, to smooth the resulting jet profile in the same way as the three-dimensional valve geometry does. Figure 2B shows the opening process of the valve model over four time steps, with the infinite (black), the zero (white), and the intermediate (gray) pressure drop areas. By adaptation of the intermediate pressure drop coefficient, the evolving jet can be adjusted to profiles measured in echocardiographic or magnetic resonance flux measurements. In the current version of this model, valve regurgitation is not included, because neither of the individuals suffered from noteworthy degrees of regurgitation across the aortic or mitral valves.

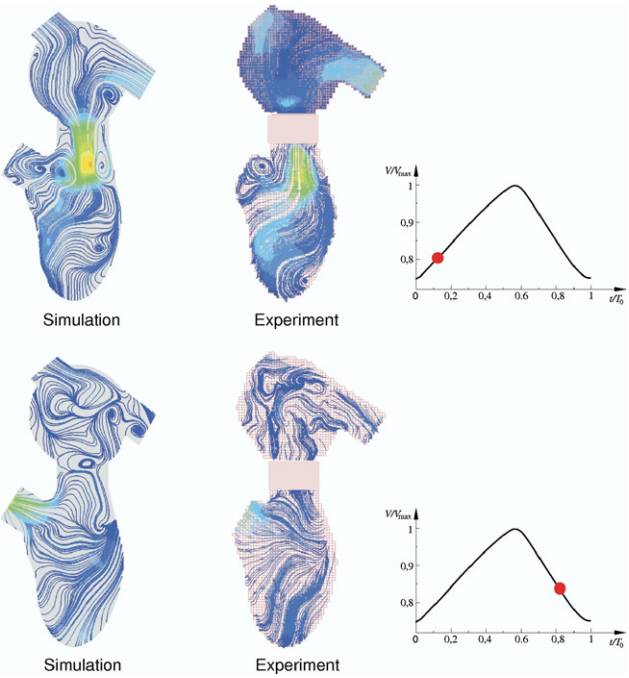

Fig 3. Comparison of simulated and directly visualized flow in a mock left ventricle and circulation model with flow patterns modeled using Karlsruhe Heart Model in the diastolic (upper panel) and systolic phase (lower panel).

Circulation Model

Boundary conditions are necessary to obtain reliable results for simulations of the human heart. To allocate these boundary conditions a model of the human circulation system is used [15]. The assignment of this KaHMo circulation model is to provide pressure boundary conditions at the venae cavae superior and inferior and at the pulmonary artery (for the right ventricle) as well as at the left atrium and the aorta (for the left ventricle). Figure 2C shows the KaHMo circulation model, which is based on the arterial body circulation model of Naujokat and Kiencke [16] and the model of Avolio [17]. The model divides the human circulatory system into elastic pipe segments. The solution of the Navier-Stokes equation for the elastic pipe flow in each segment is found by associating the electric resistance, inductivity, and capacity with the physical properties of the arterial and venous branching and the rheologic properties of blood. The flow velocity v and the pressure p correspond to the electrical current strength and voltage, respectively. The following differential equations are solved for every elastic pipe segment:

$$p_{i-1} - p_i = \frac{9 \cdot \rho \cdot l}{4 \cdot \pi^2} \cdot \frac{dv_i}{dt} + \frac{8 \cdot \mu_{eff} \cdot l}{\pi \cdot r^4} \cdot v_i = L \cdot \frac{dv_i}{dt} + R \cdot v_i$$

$$v_i - v_{i-1} = \frac{3 \cdot \pi \cdot r^3 \cdot l}{2 \cdot E \cdot d} \cdot \frac{dp_i}{dt} = C \cdot \frac{dp_i}{dt},$$

with the electric resistance R, the inductivity L, and the capacity C. E is the Young's elastic modulus of the pipe segment, l is the pipe length, r is the pipe radius, d is the wall thickness of the pipe segment, ρ is the density of the blood, and μ_{eff} is the blood viscosity.

Validation of Karlsruhe Heart Model

To validate the flow simulation by KaHMo a basic silicone model of a left ventricle was generated and equipped with two tissue valves (in aortic and mitral positions). The inflows and outflows were connected by a flexible tube (simulating the windkessel of the aorta) to create a closed circuit, which was filled with fluid containing glass spheres. This mock-left ventricle was placed in a water-filled chamber that was connected to the ventricular pumping device of a ventricular assist system

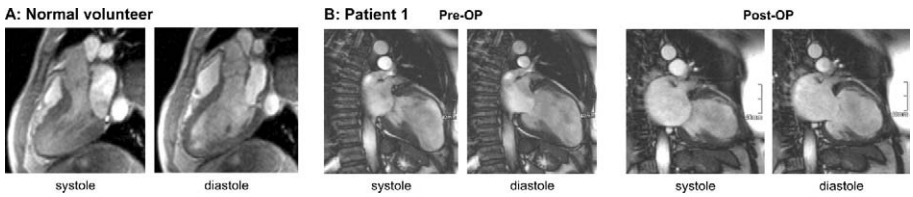

Fig 4. Long-axis cardiac magnetic resonance images of a heart from a healthy volunteer and a patient with dilated ventricle after myocardial infarction before and after surgical ventricular reconstruction. (Post-OP = postoperative; Pre-OP = preoperative.)

(Medos, Aachen, Germany) to simulate the volume changes in the mock ventricle as they occur during systole and diastole. Using a laser beam to illuminate the crystal beads inside the mock ventricle and a high-speed camera capturing the reflection of the laser light from the glass beads, it is possible to generate images of ventricular shape. The images were fed into KaHMo to simulate flow as well as directly measuring fluid vectors allowing direct flow illustration. Figure 3 shows the comparison of simulated and directly measured flow in a validation experiment in the diastolic (A) and systolic (B) phases. The illustration of flow through the mock ventricle shows a high degree of similarity between experiment and simulation, underscoring the validity of the KaHMo flow simulations [11].

Surgical Ventricular Reconstruction

Preoperative and postoperative care of the patient was performed in standard fashion. The patient was a participant of the STICH trial with ischemic cardiomyopathy and significant dilatation of the left ventricle and anterior wall akinesis. She had an indication for surgical revascularization and was randomized to coronary artery bypass graft surgery combined with SVR of the left ventricle. The procedures were performed with the patient connected to cardiopulmonary bypass and the heart arrested with cold blood cardioplegia. For the reconstruction procedure, no sizer, shaper, or patch was used. The patient had triple-vessel disease and received one arterial and one saphenous vein graft bypass. After placement of the bypass grafts, the anterior wall was incised, and the transitional zone between infarcted and viable myocardium was identified. A Fontan purse-string suture was placed and tied, and the remaining opening was closed with three overlapping felt-reinforced Prolene (Ethicon, Somerville, NJ) sutures in a 120-degree angle. The excluded scar tissue was oversewn with a felt-reinforced running 2-0 Prolene (Ethicon) suture. At the end of the procedure, the aortic cross-clamp was removed. After adequate reperfusion, the patient was weaned from cardiopulmonary bypass.

Results

Demographic and Perioperative Characteristics

Table 1 shows the demographic data of the volunteer and the patient. The volunteer was younger than the patient and except for being a smoker did not show any cardiovascular risk factors. The patient was characterized by the typical risk profile of patients with ischemic cardiomyopathy and had a previous myocardial infarction. The volunteer did not take any regular medication. The patient was on heart failure medication consistent with the guidelines. The surgical intervention was uneventful, and the patient recovered without complications.

Figure 4 shows representative end-systolic and end-diastolic CMR long-axis views of the volunteer and of the patient before and after surgery. The patient's heart is larger than the healthy heart, and surgery reduced its size. However, the postoperative shape in the patient is less spherical (ie, more ball shaped) than before surgery and than in the normal heart. Table 2 shows the functional CMR variables. As expected, the volunteer had normal ejection fraction and ventricular volume. Surgical ventricular reconstruction reduced ventricular volume by one fourth in the patient. The ejection fraction did not show a relevant increase.

Fluid Dynamics of the Healthy Human Left Ventricle

Figure 5 shows the fluid flow structure in the healthy heart during one cardiac cycle, both from two-

Table 1. Demographic Characteristics of the Healthy Volunteer and the Patient

Characteristic	Volunteer	Patient
Age (y)	35	47
Sex	Male	Female
Diabetes mellitus II	No	No
Art HTN	No	Yes
Smoker	Yes	No
History of MI	No	Yes
Renal insufficiency	No	No
Hyperlipidemia	No	Yes
CAD status	No disease	3-V disease
Mitral regurgitation	None	Trace
Medication		
Beta-blockers	No	Yes
ACE inhibitors	No	Yes
Diuretics	No	Yes
Statins	No	Yes
ASA	No	No
Coumadin	No	Yes

ACE = angiotensin-converting enzyme; Art HTN = arterial hypertension; ASA = acetylsalicylic acid, aspirin; CAD = coronary artery disease; MI = myocardial infarction; 3-V disease = three-vessel disease.

Table 2. Functional Variables of the Healthy Volunteer and the Patient Based on Cardiac Magnetic Resonance Imaging

Variable	Volunteer	Patient	
		Preoperative	Postoperative
Heart rate (bpm)	79	66	80
ESV (mL)	65	148	106
EDV (mL)	166	175	128
SV (mL)	101	27	22
EF	0.61	0.15	0.18
CO (L/min)	7.98	1.78	1.79

CO = cardiac output; EDV = end-diastolic volume; EF = ejection fraction; ESV = end-systolic volume; SV = stroke volume.

dimensional and three-dimensional perspectives as a result of the simulation. Starting with the two-dimensional flow structure, which is visualized by streamlines projected on the long-axis plane, blood is directed into the ventricle at the beginning of diastole. Owing to high differences in velocity of the inflowing blood and the blood resting inside the ventricle, a ring vortex forms behind the annular orifice of the mitral valve, which then induces a secondary vortex in the aortic outflow tract. As diastole continues, the ring vortex increases in size while moving in the direction of the ventricular apex. As a result of the cone shape of the ventricle and the inflow direction from the atrium, the ring vortex expands almost uniformly in the axial direction and asymmetrically in the radial direction. This generates a clockwise rotation of the flow, which redirects the blood toward the outlet. Finally, a three-dimensional bifurcation removes blood from the apex of the ventricle. The systole then starts with blood passing out of the ventricle into the aorta. The three-dimensional perspective in the bottom row of Figure 5 also shows the ring vortex as a consequence of the compensating motion between the different velocities and its asymmetric expansion. Although the interpretation of the flow derived from projected streamlines implies a bifurcation of the ring vortex, the three-dimensional view shows that the ring vortex leans posteriorly toward the apex of the ventricle as diastole continues. The anterior aspect of the ring vortex then dominates the clockwise rotating flow structure in the ventricle, and the posterior vortex washes out the apex. At the beginning of systole, the secondary vortex in the aortic outflow tract is washed out followed by the still-remaining vortices, which are, by this time, very low in intensity.

Effect of Postmyocardial Infarction Remodeling and Surgical Ventricular Reconstruction on Left Ventricular Fluid Dynamics

Figure 6 shows the two-dimensional projection of streamlines on the long-axis plane of the simulated hearts of the volunteer (first row) and the patient (second and third rows). In the patient, the ventricle before surgery shows the same topologic flow structure during diastole as the healthy ventricle. However, as a result of the aneurysm at the anterior apical aspect of the ventricle, the scar sags outward. This causes the intake vortex to deform asymmetrically and branch in the middle of the ventricle right after it had been formed, leading to flow losses during diastole. The outflow process is similar to that of the healthy heart.

The flow structure of the blood after surgery is completely different. The removal of the aneurysm caused an apple-shaped geometry, which significantly affected flow. The intake jet during diastole is not decelerated enough by the blood at rest and therefore generates a stagnation point flow in the apex of the ventricle. Furthermore, the ring vortex no longer expands asymmetrically. This suggests that most of the blood is not redirected toward the outlet, which then causes suboptimal outflow during systole.

Table 3 shows the fluid dynamic variables of the volunteer and the patient obtained from the simulation. M is the exchange transfusion that can be calculated from the model. It represents the remaining fractional blood volume in the ventricle after a given number of cardiac cycles n.

$$M = (1 - EF)^n$$

Although 67% of the blood in the healthy heart was replaced during the first cycle and only 2% of the initial blood volume remained in the ventricle at the end of the fourth cycle, the patient showed substantially slower washout behavior. In the patient before surgery, 35% of

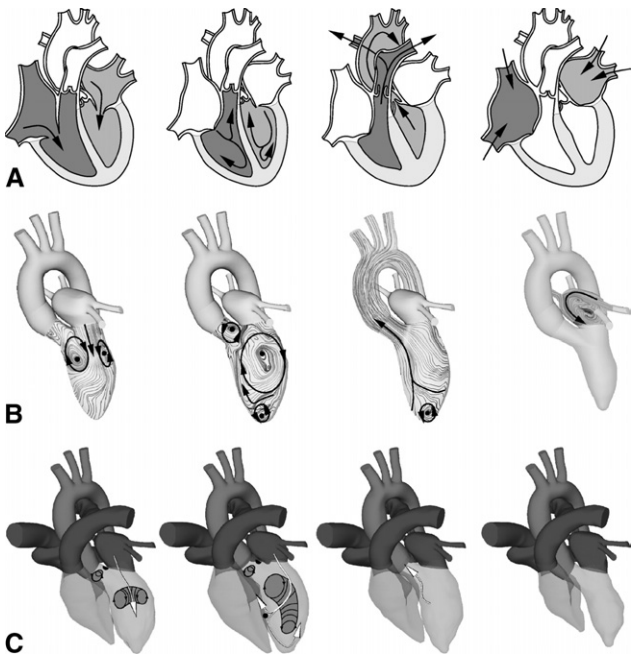

Fig 5. Schematic drawing of blood flow through a healthy heart during one cardiac cycle (A), projection of streamlines in the long-axis plane after fluid dynamic modeling with the Karlsruhe Heart Model based on data from CMR imaging (B), and three-dimensional flow interpretation (C).

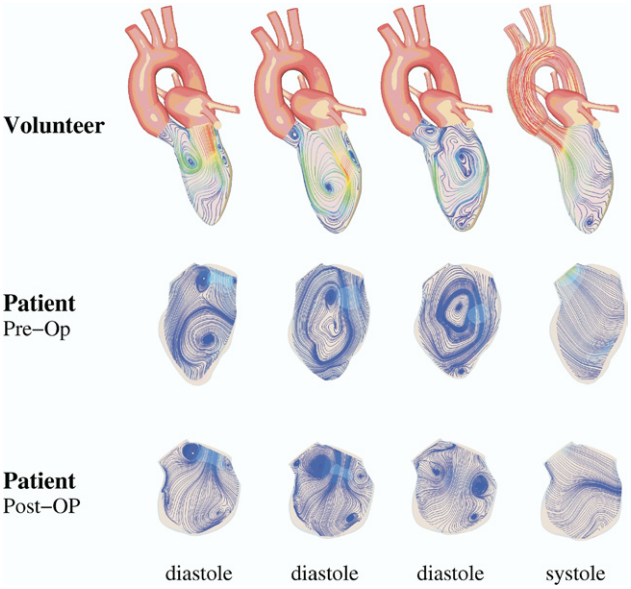

Fig 6. Karlsruhe Heart Model flow simulations in the long-axis view from the healthy volunteer (upper row) and from the patient before (middle row) and after surgery (lower row). (Post-OP = postoperative; Pre-Op = preoperative.)

the initial blood volume remained in the ventricle after four cycles. After the surgical procedure, washout was slightly lower (not significantly different) compared with before SVR despite the significant alterations in shape and fluid dynamics.

Comment

In this study we demonstrate that fluid dynamic modeling of the heart is possible based on CMR data and enables volume-independent quantitative assessment of SVR. In the future, preoperative modeling of fluid dynamics in patients undergoing SVR may help to optimize flow characteristics and potentially improve outcomes.

This is the first analysis of fluid dynamic modeling in ventricles in which the shape is surgically altered. This analysis is limited to data sets from two individuals because there are currently no tools available to perform these analyses in significant numbers. The transformation, grid generation, and modeling are based on elaborate, time-consuming steps with the need for customization of the process to each data set. The complete analysis of one data set currently requires approximately 200 working hours and must be decreased to be applicable in the clinical setting routinely.

Although limited in the number of data sets, we have generated the prospect of providing quantifiable and reproducible information about left ventricular shape and the effect of reconstructive surgery. Based on these few data sets, it appears that ball-shaped ventricles have impaired fluid dynamics. Previous reports focused on global functional variables such as the ejection fraction or volume [4, 18]. Although all of these variables are important and valuable, it is difficult to infer any information from them with respect to the influence of shape. Most descriptions on the impact of shape on function are experimental or rely on assumptions that have not been validated in human diseased hearts. For example, the notion that myofiber orientation changes within a regional segment of myocardium (eg, in the basolateral wall) with the development of dilatation in response to an anterior infarction has never been demonstrated in vivo [19]. Similarly important, the impact of a reconstruction procedure on local fiber orientation is also not known. These efforts are further complicated by the fact that the best clinical results of SVR come from a surgeon who is most likely to create the shapes that are considered least favorable (ie, ball shapes) [4]. Our findings of similar fluid washout in the ball-shaped ventricle of the patient after surgery despite changes in ejection fraction, shape, and fluid dynamics may explain the fact that patients after SVR are not at greater risk for thrombus formation than before. However, the analysis of one patient only allows us to speculate in this direction. More analyses are certainly necessary to answer these questions.

Nevertheless, we are presenting a three-dimensional numeric model of the human heart. The model is based on clinical magnetic resonance imaging measurements and can therefore be used on a patient-specific basis without subjecting the patient to additional diagnostic procedures. The field information available from the simulation could be used to calculate quantities that cannot yet be assessed in vivo, such as shear stresses and hemodynamic losses, and to derive variables that can be used to evaluate the function of the ventricle. These variables may be used as indicators for the impact of the surgical procedure on outcome from a fluid mechanical point of view, and may therefore help the surgeon to decide in the future whether the planned intervention will have the desired effect. One of the key points for such a method to become routinely established is its validation.

We demonstrate here that KaHMo is able to accurately predict flow in a mock model of a left ventricle (Fig 3). In a previous study, the KaHMo had been

Table 3. Fluid Dynamic Variables of the Healthy Volunteer and the Patient

		Patient	
Variable	Volunteer	Preoperative	Postoperative
T_0	0.76 s	0.91 s	0.83 s
M cycle (1st)	33%	82%	80%
M cycle (2nd)	16%	53%	64%
M cycle (3rd)	7%	44%	50%
M cycle (4th)	2%	35%	39%
Wo (sys)	27	21	20
Re (sys)	4150	819	765

M = exchange transfusion; Re = Reynolds number; sys = systolic; T_0 = heart cycle; Wo = Womersley number.

validated by an overall comparison between the simulated data and velocity data obtained by additional flux measurement. It had been shown that the model is capable of predicting a realistic flow field in the human ventricle. Furthermore, this study showed that the intraventricular flow pattern is sensible to a correct representation of the atrial geometry [11]. In the present study, the magnetic resonance imaging data sets of the patient's heart did not include any atrial information, but a generic vessel was used. The representation of the valves used in the current study does not include a potential effect of the valve leaflets on flow. Nonetheless, the validation shows no discrepancy in the valve area. This justifies the assumption that the healthy mitral and tricuspid valve leaflet does indeed orient itself in the flow and does not steer the flow. However, both validation and valve function will have to be included in the model under practically relevant conditions for the model to be used to its maximal potential. In this study, the patient did not suffer from valvular regurgitation or stenosis, so that the lack of modeling for valvular regurgitation did not affect the results.

The presented analysis describes an attractive tool to assess the fluid patterns in the heart in patients with ischemic heart disease. It is conceivable for the future to simulate the optimal shape for each patient before surgery, so that the surgeon knows what shape would be optimal for the patient before the procedure is performed. Although this vision is currently being investigated, the generation of the current fluid models for each patient is still very cumbersome and time-consuming and has to be much improved.

Imaging flow through the ventricle has also been performed by magnetic resonance phase-contrast velocity mapping [20]. This procedure allows the direct visualization of flow without the need to transform the data for model feeding. Although this method has not been applied in patients undergoing surgery, the differences in flow patterns and flow efficiencies between patients with dilated cardiomyopathy and normal subjects were similarly impressive. Although the magnetic resonance analysis is not as precise in the illustration of flow patterns, it has definite strengths in the quantification of energy losses and efficiency calculations. Time will tell whether the laborious nature of our simulation can be simplified enough to withstand this competition.

Irrespective of any competition, our model has not been developed purely for clinical application. Others have described mathematical models of the heart. Focus has been placed on assessing the relation between pump function and myofiber mechanics [21, 22] or the development of algorithms to describe left ventricular geometry [23]. All of these models have the potential to be applicable to clinical practice in the future, but none have contributed thus far. We have recently begun to integrate the influence of regional function and myofiber orientation into our attempts to model the heart and the influence of disease and its treatment (unpublished observations, 2008).

In conclusion, we have demonstrated that fluid dynamic modeling of the heart is possible based on CMR data and enables volume-independent quantitative assessment of SVR. In the future, preoperative modeling fluid dynamics in patients undergoing SVR may help to optimize flow characteristics and potentially improve outcomes.

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